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Analysis of Clip-induced Ischemic Complication of Anterior Choroidal Artery Aneurysms

Objective : The surgical approach is typically similar to those used for other supraclinoid internal carotid artery (ICA) lesions. However, the surgical clipping of this aneurysm is complicated and as a result, can result in postoperative ischemic complications. We studied to clarify the clip-induced ischemic complication risk of AChA aneurysm and to get the benefits for helping decision making.

Methods : We retrospectively investigated 53 cases (4.0%) of AChA aneurysm treated surgically. We divided the AChA aneurysm to 3 subtype according to the origin of aneurysmal neck; A type originating from the AChA itself, J type from junction of AChA and ICA, and I type from the ICA itself. We evaluated brain CT about 1 week post-operative day to confirm the low density in AChA territory.

Results : Ruptured aneurysm was 26 cases and unruptured aneurysm 27 cases. The aneurysmal subtype of A, J, and I was 13, 17, and 23 cases. Of the 53 cases who performed surgical neck clipping, twelve (22.6%) had postoperative AChA distribution infarcts. Increased infarct after neck clipping had statistic significance in non-I subtype ($p=0.005$).

Conclusion : It is easy to classify as "easy" surgery. But surgery for AChA aneurysms carries with it a significant risk of postoperative stroke. Don't always stick to clipping only, especially in non-I type of incidental small aneurysm, which has high risk of post-clip ischemic complications.

KEY WORDS : Anterior choroidal artery infarction · Clip · Intracranial aneurysm.

INTRODUCTION

Anterior choroidal artery (AChA) aneurysms account for 2 to 5% of all intracranial aneurysms^{2,5,11}. The surgical approach and methods for AChA aneurysms are typically similar to those used for other supraclinoid carotid artery lesions^{6,8,11}. However, because of the parenchymal area supplied by the AChA, the surgical clipping of this aneurysm is complicated and as a result, can result in postoperative ischemic complications in 5 to 50% of cases. Occlusion of the AChA sometimes causes significant hemiparesis, hemianesthesia, or hemianopsia, which is referred to as AChA syndrome¹. AChA syndrome occurs frequently and is one of the most serious complications of the surgery for AChA aneurysms¹⁰. The problem with AChA aneurysms is the increased detection rate of unruptured asymptomatic ones, especially when coincidentally found during an operation. This is more likely to result in debilitating postoperative ischemic complications. To improve our surgical approach to minimize the risk associated with AChA aneurysms, we reviewed our institutional surgical experience with 53 patients to identify factors contributing to a high risk of postoperative stroke.

MATERIALS AND METHODS

We retrospectively investigated 53 cases of AChA aneurysm out of 1315 intracranial aneurysms, which were treated surgically by same operator at a single institute between 1984 and 2005. For each patient, we recorded demographic data, clinical presentation, aneurysm size and anatomical type, presence of other arterial aneurysms and presence or absence of AChA aneurysm rupture. In this retrospective study, AChA aneurysms were defined as those arising from the supraclinoid ICA in close proximity to the origin of the AChA, or those arising from the AChA itself. We characterized the aneurysms as A, J or I type. A type is the sac formed on the trunk of the AChA itself (Fig. 1). J type is the sac formed in both the

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Fig. 1. A type : aneurysm (asterisk) originating entirely from the anterior choroidal artery itself.

Fig. 2. J type : sac (asterisk) formed in part from the anterior choroidal artery and internal carotid artery.

Fig. 3. I type : aneurysm (asterisk) originating from the internal carotid artery, near or adjacent to the origin of the anterior choroidal artery.

Fig. 4. Computed tomography scan 1 week after clipping of left anterior choroidal artery aneurysm showing low density (arrow head) on genu of internal capsule.

the AChA (Fig. 4). We excluded patients who had untreated aneurysms or aneurysms that were treated by means other than clipping.

RESULTS

In cases with a ruptured aneurysm, we identified an infarction in 5 out of 26 cases (19.2%). On the other hand, 7 of 27 unruptured aneurysm cases (25.9%) were associated with infarction. While the prevalence of post-clip infarction seems greater with unruptured versus ruptured cases, the difference was not statistically significant ($p=0.056$), as shown in Table 1. The sac size was less than 5 mm in 23 cases and of these, infarction occurred in 7 cases (30.4%). From the 30 cases with a sac size greater than 5 mm, 5 (16.7%) developed infarctions. But, the difference in infarction incidence between large and small sacs was not statistically significant ($p=0.235$), as shown in Table 2. The frequency of infarct was the highest among the J type aneurysms, followed by A and I types respectively. Non-I (A and J) types

had a greater incidence of post-clip infarct than I type, and this difference was statistically significant ($p=0.005$), as shown in Table 3. Unruptured small aneurysm less than 5 mm had a higher prevalence of post-clip infarction, but it was not statistically significant. Further, non-I type (36.7%) aneurysms were more vulnerable to post-clip infarction than I type aneurysms (4.4%).

DISCUSSION

The anterior choroidal artery (AChA) is a very small, but important branch of the internal carotid artery. The AChA arises mainly from the posterolateral aspect of the internal

Table 1. Aneurysmal rupture and postoperative infarction

Rupture (n)	Infarction	
	Yes	Incidence (%)
Yes (26)	5	19.2
No (27)	7*	25.9

* $p=0.560$

Table 2. Aneurysmal size and postoperative infarction

Size (mm), (n)	Infarction	
	Yes	Incidence (%)
<5 (23)	7*	30.4
$5 \leq 10$ (26)	4	15.4
≥ 10 (4)	1	25

* $p=0.235$

Table 3. Aneurysmal type and postoperative infarction

Type (n)	Infarction	
	Yes	Incidence (%)
A (13)	3	23.1
J (17)	8	47.1
I (23)	1*	4.3

* $p=0.005$. A : aneurysm originating entirely from the AChA (anterior choroidal artery) itself, J : sac formed in part from the AChA and ICA (internal carotid artery), I : aneurysms originate from the ICA, near or adjacent to the origin of the AChA

carotid artery about 3.2 mm distal to the posterior communicating artery and 5.2 to 5.6 mm proximal to the carotid bifurcation. The AChA supplies penetrating branches from its proximal segment to the optic tract and to the medial segment of the globus pallidus. As the artery courses posteriorly, it branches off to the uncus, piriform cortex, amygdala, and the anterior hippocampus and dentate gyrus, and it supplies the middle third of the cerebral peduncle, the subthalamus, and the ventral anterior, ventral lateral, pulvinar, and reticular nuclei of the thalamus. This artery also gives off penetrating branches at the level of the lateral geniculate body that supplies the posterior two thirds of the posterior limb of the internal capsule together with its retrolenticular segment, including the origin of the optic and auditory radiation and the tail of the caudate nucleus. The anterior half of the lateral geniculate body is also supplied by the AChA, which terminates in the choroid plexus. There are many variations and anastomoses associated with the middle cerebral artery, the PComA, and the posterior cerebral artery, and the most constant branches include the optic tract, cerebral peduncle, posterior limb of the internal capsule and choroid plexus^{7,12}.

AChA syndrome was first described in 1925 by Foix et al. includes hemiplegia, hemianesthesia and hemianopsia. Yasargil et al. and Suzuki et al. reported a patient who had suffered from AChA syndrome after the clipping of an ICA-AChA aneurysm, where the AChA was thought to be preserved^{9,12}. Helgason reported a patient with this syndrome caused by vasospasm after SAH⁴.

The causes of postoperative AChA syndrome are classified into 'during the operation' and 'post-operation'. Due to excessive manipulation of vessels during an operation, AChA syndrome can result when the AChA itself or its branches or perforators clipped with it form a thrombus. Because of the many variations in the branches and perforators of the AChA, cerebral ischemia occurs even when blood flow had been confirmed after the clipping³. Immediate postoperative AChA syndrome occurred when the AChA became entangled with the clip; cerebral ischemia occurred as a latent response after the operation, vasospasm developed in the AChA and the collateral blood flow reduced after the operation⁹. In this study, when the aneurysm was less than 5 mm, unruptured, and non-type I, the incidence of cerebral ischemia increased after clipping. The etiology of this outcome was not clearly identified, but several possibilities were considered. As the size of the aneurysm gets smaller, the artery is decompressed by the clip and its diameter is reduced. It was unexpected that cerebral ischemia would be more common in the case of non-ruptured aneurysms rather than the case of the ruptured one. This is caused

by the higher incidence of vasospasm in ruptured cases. The opposite outcome occurred when a small unruptured aneurysm of AChA was accidentally found during the operation for another ruptured cerebral aneurysm and many of them were clipped. In case of non-type I aneurysms, cerebral ischemia was commonly due to vessel strangulation by the clip or a decrease of the vascular diameter and the higher possibility of ligation of the peripheral vascular branches than in type I. Friedman et al. reported that the cerebral ischemia after the clipping showed a higher incidence than in other cases, which was 44%, for type I cases³. Also, it was reported that the small and accidentally found aneurysm that was located in this area was unlikely to have the clipping.

In cases where cerebral ischemia occurred in the region of AChA after the operation, the strategy of the treatment depends on the etiology. If AChA syndrome was identified immediately after the operation, it might be that the AChA is interrupted by a clip or the artery is entangled, which means another clipping by reoperation should be considered after the confirmation by angiography. In this case, a good outcome is expected if rapid management is performed, but it is rare that the symptoms are improved right after the reoperation. In the case of the latent cerebral ischemia, vasospasm and reduced lateral blood circulation are suspected, so it is helpful to maintain hypertension and hypervolemia.

In this study, in cases of non-Type I aneurysms of the AChA, the incidence of cerebral ischemia was high after the clipping. The etiology of this outcome could possible involve vascular strangulation or the low incidence of clipping of the AChA itself and relatively low manipulation. In cases of non-Type I aneurysms of the AChA, based on the preoperative angiography or on the manifestations during the operation, other methods such as wrapping might be helpful for reducing postoperative ischemic complications.

CONCLUSION

For aneurysms of the AChA, the operation is not difficult due to easy access, just as for other supraclinoid aneurysms, but the risk of the postoperative ischemic complications is higher for aneurysms of the AChA. Especially for aneurysms that are not Type I, less than 5 mm and non-ruptured, there is an increased incidence of cerebral ischemia in the region of AChA. It is important to perform the operation with care, confirming the variations of the AChA during the operation. Further, clipping or other methods such as wrapping are not recommended due to the anatomical characteristics of the AChA that can lead to a higher incidence of ischemic complications.

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